

60th Annual Scientific Session & Expo

E334

JACC April 5, 2011

Volume 57, Issue 17



CARDIAC FUNCTION AND HEART FAILURE

RELATIONSHIP BETWEEN VASCULAR FUNCTION, VENTRICULAR-VASCULAR COUPLING AND PERIPHERAL PRESSURE PULSATILITY IN HYPERTROPHIC CARDIOMYOPATHY

ACC Poster Contributions

Ernest N. Morial Convention Center, Hall F

Monday, April 04, 2011, 3:30 p.m.-4:45 p.m.

Session Title: Cardiomyopathies/Myocarditis/Pericardial Disease

Abstract Category: 22. Cardiomyopathies/Myocarditis/Pericardial Disease

Session-Poster Board Number: 1119-25

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Background: Patients with hypertrophic cardiomyopathy (HCM) have coronary and peripheral conduit vascular dysfunction. Interestingly, peripheral microvascular function is preserved in patients with HCM. The etiology for this heterogeneity between vascular beds in vascular function in HCM is unclear. Therefore, we examined the role of ventricular-vascular coupling (VVC), pressure pulsatility, and microvascular function in a HCM population.

Methods: We studied 50 patients with HCM (without left ventricular (LV) outflow tract obstruction at rest or during exercise) and 50 control subjects (without HCM devoid of coronary artery disease, CAD-), matched for age, height, weight, gender, blood pressure, heart rate, and other cardiovascular risk factors (i.e. diabetes mellitus, dyslipidemia, smoking). Forty-four patients without HCM with CAD+ served as positive controls for vascular dysfunction. Pulse volume waves were measured using finger plethysmography (peripheral artery tonometry, PAT) and used to calculate augmentation index (Alx, a measure of VVC) and the reactive hyperemia index (RHI, a measure of microvascular function). Pulsatility was calculated as pulse pressure / mean arterial pressure.

Results: PAT-RHI was preserved in HCM (2.07 ± 1.0 vs. $2.16 \pm 0.09\%$, HCM vs. CAD- controls respectively, $p = 0.787$). Both HCM and CAD- had higher PAT-RHI than CAD+ ($1.69 \pm 0.08\%$, $p < 0.05$). PAT-Alx was similar between HCM and CAD- controls (5.6 ± 3.0 vs. $6.3 \pm 2.8\%$, $p = 0.985$). Both HCM and CAD- had lower PAT-Alx than CAD+ ($18.6 \pm 2.9\%$, $p < 0.05$). Pulsatility was similar between HCM and CAD- controls (0.53 ± 0.02 vs. 0.55 ± 0.02 , $p = 0.655$). Both HCM and CAD- patients had lower pulsatility than CAD+ patients (0.64 ± 0.02 , $p < 0.05$).

Conclusions: In addition to preserved microvascular function, our findings suggest that patients with HCM have preserved VVC and low peripheral pressure pulsatility. VVC in HCM may attenuate the propagation of potentially deleterious pulsatile pressure into the peripheral vasculature, which may, in turn, stave downstream vascular damage and preserve microvascular function in HCM.